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NEUROSPECIFIC ENOLASE – EARLY DIAGNOSTICAL MARKER OF CNS DAMAGE

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Mechanisms of hypoxic damage of brain cells are characterized by a complex cascade of pathophysiological processes. Final result of this mechanism is the death of neurons due to necrosis and apoptosis. In newborn babies is not always possible to objectively assess the condition of the CNS defeat, because very often the severity of lesions does not correspond to clinical symptoms, especially in premature newborns. So far determination the severity of hypoxic-ischemic CNS lesions is still very actually in modern medicine. More objective method of such an assessment is determining the activity of neurospecific enolase (NSE).

Research purpose: to increase the efficiency of diagnosis of hypoxic-ischemic CNS lesions in premature infants by determining the activity of NSE in early neonatal period.

The concentration of NSE was determined in 15 conventionally healthy preterm infants (CHPI), which made the comparison group and 64 premature babies with hypoxic-ischemic CNS lesions on the 1-7 day of life. They were divided into three groups: I group – 26 premature children with mild CNS lesions; II group – 20 premature children with severe hypoxic-ischemic lesions and low birth weight; III group – 18 premature newborns with severe damage of central nervous system and extremely low birth weight.

Determining the level of NSE in serum of premature infants found that at the end of the early neonatal period in brain cells of children with hypoxic-ischemic CNS lesions showed destructive changes of neuronal membranes. About this evidenced significant increase the level of enzyme. So, if perinatal CNS lesions of mild degree occur, NSE content in the blood of children of I group increased by 45% relative to the comparison group ($p < 0,05$). Thus, even mild hypoxia caused a significant alteration of neuronal membranes and damage brain tissue. In the second group of infants with low birth weight on the base of severe hypoxia there was further increase activity of this enzyme in the blood, which manifested by increased serum concentrations of NSE in 2,2 times relative to the children of I group ($p < 0,001$). It should also be noted that its activity in case of severe hypoxia was almost 3,3 times higher relative to the comparison group ($p < 0,001$).

Maximum concentration of enolase reached in premature infants with very low birth weight and severe hypoxic-ischemic injury of the CNS. Its contents in serum of premature neonates of III group was 4 times greater than in comparison group ($p < 0,001$), increased 2,9 ($p < 0,001$), and 1,3 ($p < 0,05$) times relative to infants of I and II groups, respectively.

Thus, hypoxic-ischemic injury of the nervous tissue causes increased permeability of cell membranes and leave into the blood such neurospecific protein as NSE. The high rates of NSE in serum of premature infants on a base of hypoxic injury describe breach of the functional condition of cell membranes of neurons and correspond to the severity of brain damage due to hypoxia. Therefore, to assess the severity of hypoxia is necessary to determine the level of NSE in serum in the early neonatal period in premature infants.

EVALUATING THE EFFECTIVENESS OF INHALED STEROIDS IN CHILDREN WITH BRONCHIAL ASTHMA

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Properties of inhaled steroids are fundamentally different from the system hormones: they work mainly locally on the bronchial mucosa; they are in low quantity getting into the bloodstream and minimally affect the whole body. Patients don’t get used to inhaled hormones, on the contrary from systemic. More common steroids for inhalation are budesonide, beclomethasone, fluticasone, ciclesonide, flunisolide etc.